BRITISH MEDICAL JOURNAL

LONDON, SATURDAY 9 NOVEMBER 1985

Recent changes in dental caries

In the past 10 to 15 years dental caries has declined by 35-50% among children in most industrialised countries.[1,2] Not only have caries scores declined (expressed as the number of decayed, missing, and filled teeth or surfaces) but the proportion of children free of caries has increased. For example, in one survey in Devon on 12 year old children it rose from 5% in 1971 to almost 22% in 1981.[3] Most of the data refer to children, but similar trends have been reported in young adults in Denmark[4] and in servicemen up to the age of 40 in the United States.[5] The reverse trend is apparent in developing countries—presumably rising standards of living have led to increased sugar intake.[6]

This reduction in caries has led to a lot of speculation, but the cause seems likely to be as multifactorial as the disease itself,[7] and probably different factors have operated in different countries.

One possibility is that changes have occurred in diagnostic criteria over this period, particularly as in only a few of the surveys were the early caries scores collected by the same observers as those responsible for the recent figures. Evidence has accumulated recently that some very early lesions ("white spots"—that is, lesions without cavitation or a break in the enamel) may not develop into true cavities but either become arrested or may even recede by remineralisation encouraged by exposure to fluoride.[8,9] The New Zealand School Dental Service recognised this process and in 1976 adopted new standards which specified that fillings should be confined to those lesions with a visible break in the enamel and affecting dentine.[10] Though no other country is known formally to have changed its diagnostic criteria for caries, the concept that early lesions may not require fillings is widely known. Possibly, therefore, many early lesions that would have been filled a decade or two ago are now not even recorded. Nevertheless, some of the surveys showing a fall in caries were carried out with highly standardised criteria, and in these cases at least the fall is unlikely to be an artefact.[11] The reported changes seem to represent a real decrease in both the prevalence and severity of dental caries.

Among the more speculative explanations for this decrease are that the organisms responsible for caries have become less virulent or that the widespread use of antibiotics has had a prolonged effect on oral bacteria.[12] A more reasonable explanation—also lacking firm evidence—is that the amount and frequency of sugar consumption among children have decreased. Small reductions in the overall sale of sucrose have been reported in some countries, but this has been compensated by an increase in the consumption of corn syrup in manufactured products.[13] The limited evidence suggests that this is only marginally less cariogenic than sucrose. Data showing a fall in the household purchase of sugar ignore the large amount ("hidden sugar") in so many manufactured foods.[14] Little is known about the distribution of sugar within the family, but a recent dietary survey of 405 British schoolchildren aged 11-13 reported an average daily intake of 118 g—close to the average intake of the population as a whole and giving no indication of a lower intake by children.[15] The use of feeders and sweetened dummies has been strongly discouraged, and sugar is no longer added to bottle feeds, so caries resulting from their use is presumably less; but this represents only a small part of caries in the population as a whole. The type of confectionery eaten by the older children has also changed—more chocolate covered bars, probably eaten at once, in place of sweets eaten individually over a longer time[16]—but the effect on caries is uncertain.

Another possibility is that dental health education, including advertising toothpastes on television, has made the public more conscious of the possibility of preventing caries, leading to more frequent and more thorough toothbrushing, and this is supported by the steady increase in the sale of toothpastes. Surveys relating the frequency of toothbrushing to caries suggest, however, that increased toothbrushing could not in itself have reduced caries to the extent recently observed.[17] Some effect by these factors cannot be dismissed, but the limited evidence of their potential importance suggests that at most their influence could account for only a small part of the observed change.

In some countries (for example, the United States and New Zealand) the widespread fluoridation of water probably played a large part in reducing caries. In Scandinavia, where water is not fluoridated, the large scale programmes of fluoride rinses and tablets have been effective.[18] The reduction has also occurred, however, in parts of Britain which do not receive fluoridated water[3] and in the Netherlands, where fluoridation has been stopped.[12] The factor common to all the countries in which a fall in caries has been reported is the near universal use of fluoridated toothpastes, and this seems the most likely cause in most of Britain and countries lacking other sources of fluoride. Further circumstantial evidence for the role of fluoride toothpastes is that in France their use has lagged behind that of other European countries, and the fall in caries has been much smaller[2]; in

Japan, where they are almost unknown, caries has not changed at all.[2] Nearly 100 clinical trials have been reported on fluoride toothpastes with mean reductions in caries ranging from zero to 50%[19] and an overall mean of 24%. The effect has been higher on teeth erupting during the trial than on teeth present when the trial began.[20 21] Newly erupted teeth are incompletely mineralised and are both more susceptible to the damaging effect of sugars[22] and better able to benefit from the caries preventing action of fluoride.[20 21]

At first sight, the recent reduction seems larger than could be accounted for by the use of fluoride toothpastes. In the clinical trials, usually lasting three years and on children 12-15 years old, only eight to 12 teeth will erupt during the trial and receive the maximum benefit. In general use, however, children will be applying the toothpaste from the earliest age that they can use a toothbrush and all their permanent teeth will be in contact with fluoride immediately after eruption, thus increasing the average reduction. Furthermore, children aged under 5 are unlikely to spit out all their toothpaste, so that it remains in contact with the teeth for a longer time than occurs with older children, who usually do spit and rinse their mouths after toothbrushing.[23] The effects of avoiding rinsing and delaying spitting out after using fluoride toothpastes on the concentration of fluoride in the saliva and the duration of its contact with the teeth have recently been measured.[24] The results confirm that delays in spitting out and rinsing (as occurs with young children) might increase the effectiveness of these toothpastes in reducing caries. Finally, the swallowing of residual fluoride may increase systemically the resistance to caries of developing permanent teeth.

The evidence strongly suggests, then, that fluoride toothpastes have been the main factor in the reduction of caries in Britain, where only about 10% of the population receive fluoridated water. Fluoride toothpastes are unusual in that their unrestricted use by the public seems to have made a greater impact than had been predicted from the results of well controlled clinical trials.

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1 Glass RL, ed. The declining prevalence of dental caries. *J Dent Res* 1982;**61**:1304-83.
2 Renson EC (and members of a working group). Changing patterns of oral health and implications for oral health manpower. *Int Dent J* 1985;**35**:235-51.
3 Anderson RJ, Bradnock G, Beal JF, James PMC. The reduction of dental caries prevalence in English schoolchildren. *J Dent Res* 1982;**61**:1311-6.
4 Fejerskov O, Antrolt P, Gadegaard E. Decrease in caries experience in Danish children and young adults in the 1970s. *J Dent Res* 1982;**61**:1305-10.
5 Howard JH, Young JM, Rake A, Medina R, Alanis PJ. Reduction in DMFT and DMFS scores between 1977 and 1982 USAF military populations. *J Dent Res* 1984;**63**:182 (abstract 105).
6 Barmes DE. Indicators for oral health and their implications for developing countries. *Int Dent J* 1982;**33**:60-6.
7 McEniery TM, Davies GN. Brisbane dental survey 1977: a comparative study of caries experience of children in Brisbane, Australia, over a 20-year period. *Community Dent Oral Epidemiol* 1979;7:42-50.
8 Backer-Dirks O. Posteruptive changes in dental enamel. *J Dent Res* 1966;**45**:503-11.
9 von der Fehr FJ, Loe H, Theilade E. Experimental caries in man. *Caries Res* 1970;**4**:131-48.
10 de Liefde B, Ritchie GR. Evaluation in dental public health in New Zealand. *NZ Dent J* 1984;**80**:8-14.
11 Todd JE, Dodd T. *Children's dental health in the United Kingdom*. London: Office of Population Censuses and Surveys, HMSO, 1985.
12 Loesche WJ, Eklund SA, Burt BA. Relationship between antibiotic use and DMF scores in children. *J Dent Res* 1982;**61**:abstract 225, abstract 420.
13 De Paolo DF, Soparkar PM, Tavares M, Allukian M Jr, Peterson H. A dental survey of Massachusetts schoolchildren. *J Dent Res* 1982;**61**:1356-60.
14 Jarvis PN, Lennon MA. Sugar consumption in Britain. *J Dent Res* 1983;**62**:439 (abstract 222).
15 Hackett AF, Rugg-Gunn AJ, Appleton DR, Eastoe JE, Jenkins GN. A 2-year longitudinal nutritional survey of 405 Northumberland children initially aged 11·5 years. *Br J Nutr* 1984;**51**:67-75.
16 The Cocoa Chocolate and Confectionary Alliance. *Annual report 1984*. London: Cocoa Chocolate and Confectionary Alliance, 1985.
17 Bibby BG. Do we tell the truth about preventing caries? *Journal of Dentistry for Children* 1966;**33**:269-79.
18 Koch G. Evidence for declining caries prevalence in Sweden. *J Dent Res* 1962;**61**:1340-5.
19 Murray JJ, Rugg-Gunn AJ. Fluoride toothpaste and dental caries. *Fluorides in caries prevention*. Bristol: Wright, 1982: 100-26.
20 Naylor MN, Emslie RD. Clinical testing of stannous fluoride and sodium monofluorophosphate dentifrices in London schoolchildren. *Br Dent J* 1967;**126**:17-23.
21 Tucker GJ, Andlaw RJ, Burchell CK. The relationship between oral hygiene and dental caries incidence in 11-year-old children. *Br Dent J* 1976;**141**:75-9.
22 Miller CD. The dental caries response to rats fed cariogenic and non-cariogenic diets for different periods of time. *J Nutr* 1958;**66**:113-21.
23 Barnhart WE, Hiller LK, Leonard GT, Michaels SE. Dentifrice usage and ingestion among four age groups. *J Dent Res* 1974;**53**:1307-13.
24 Collins WJN, Weetman DA, Stephen KW, Smalls MJ. Salivary F concentrations following toothbrushing [Abstract]. *Caries Res* 1984;**18**:155.

Hyperplastic gastropathy

It is 94 years since Menetrier described the condition, hyperplastic gastropathy, that still carries his name. The confusion that has surrounded his eponymous disease is witnessed by a terminological pot-pourri of synonyms.[1 2] While giant gastric mucosal folds may be due to carcinoma, lymphoma, or other less common conditions, they are the cardinal feature of primary hyperplastic gastropathy.[1 3] This term was introduced by Ming, who classified it pathologically into mucous cell hyperplasia, glandular cell hyperplasia, or mixed mucous-glandular cell hyperplasia.[4] Menetrier's disease should now be considered as a well defined condition within the spectrum of hyperplastic gastropathy corresponding to Ming's mucous cell type. It has four distinct features: large gastric mucosal folds, low or less commonly normal gastric acid secretion, increased gastric protein loss, and characteristic histological appearances. The microscopical features include foveolar hyperplasia, atrophy of the fundic glands, and pseudopyloric metaplasia. Cystic change and displacement of epithelium within the submucosa are common. Macroscopically the gastric mucosa is thrown into extravagant folds 3-4 cm high. The condition may affect the whole stomach but is usually localised to the body, characteristically sparing the antrum.

At the opposite end of the spectrum of hyperplastic gastropathy lies Schindler's disease, or hypersecretory hyperplastic gastropathy.[5] This corresponds to Ming's glandular cell type and is characterised by large gastric mucosal folds, normal or high gastric acid secretion, normal protein metabolism, and parietal and chief cell hyperplasia with a relative or absolute reduction in mucous cells. It should be emphasised that Menetrier and Schindler's diseases represent the two extremes of the spectrum of hyperplastic gastropathy with intervening mixed forms (Ming's mixed mucous-glandular cell type). Fieber and Rickert's analysis of 50 well documented case reports of hyperplastic gastropathy shows that Menetrier's disease is the commonest type (66%), followed by Schindler's disease (22%) and the mixed type (12%).[2]

While there is some evidence that Schindler's disease may progress to Menetrier's disease and then on to atrophic gastritis, other authorities deny such progressive change. Spontaneous regression has been documented.

Hyperplastic gastropathy is over three times more common in men than women, and the average age at diagnosis is 40.[2] There is a self limiting, protein losing gastropathy of children, but its transient course, allergic background, and association with viral illness (especially cytomegalovirus) mean that it is unlikely to be related to adult Menetrier's disease.[6]

Theories outnumber facts about the cause of hypertrophic gastropathy.[1 2] Two popular theories have been the trophic effect of gastrin and irritation due to duodenogastric reflux. Nevertheless, out of 14 reported cases, the serum gastrin